UNITED STATES DISTRICT COURT
DISTRICT OF MASSACHUSETTS

DANA-FARBER CANCER INSTITUTE, INC.,	)	
	)	
Plaintiff,	)	
	)	
v.	)	
	)	
GATEKEEPER PHARMACEUTICALS, INC.,	)	
	)	
Defendant.	)	CIVIL ACTION NO.
	)	10-11613-DPW
	)	
GATEKEEPER PHARMACEUTICALS, INC.,	)	
	)	
Counter-Claimant,	)	
Third-Party	)	
Plaintiff,	)	
	)	
v.	)	
	)	
DANA-FARBER CANCER INSTITUTE, INC.,	)	
	)	
Counter-Defendant,	)	
	)	
and	)	
	)	
NOVARTIS PHARMA, A.G.; NOVARTIS INTERNATIONAL PHARMACEUTICAL, LTD.; and NOVARTIS INSTITUTES FOR BIOMEDICAL RESEARCH, INC.,	)	
	)	
Third-Party	)	
Defendants.	)	

MEMORANDUM AND ORDER
October 12, 2012

Dana-Farber Cancer Institute, Inc. brought this declaratory judgment action against Gatekeeper Pharmaceuticals, Inc. to establish the rights and responsibilities of each party in connection with a newly discovered compound, WZ-4-002, which has the potential to be an effective treatment for a subset of non-

small-cell lung cancers that are resistant to two other existing drug treatments. The actions and choices by the parties in this litigation made many of the issues moot, and have narrowed the focus of the case to whether third-party defendants affiliated with the Novartis pharmaceutical group were entitled to license WZ-4-002 under the Collaborative Research Agreement (the "Research Agreement") between Novartis and Dana-Farber.

The Novartis third-party defendants filed a motion for summary judgment on Gatekeeper's claim of intentional interference with contractual relations. Gatekeeper filed a cross-motion for summary judgment. I have denied Novartis's motion for summary judgment as to Gatekeeper's claim of intentional interference with contractual relations and have granted Gatekeeper's cross-motion. This Memorandum provides the extended explanation for my actions on these motions and a direction to the parties to develop a schedule for the damages phase of this litigation.

I. BACKGROUND

A. The Parties

Plaintiff Dana-Farber is the not-for-profit cancer research corporation that employed the scientists who discovered WZ-4-002. Its principal place of business is in Boston, Massachusetts. Dana-Farber receives funding from a variety of sources, including Novartis Institutes for Biomedical Research, Inc.

Defendant, counter-claimant, and third-party plaintiff Gatekeeper is a Delaware corporation with its principal place of business in Millbrae, California. Gatekeeper was founded by the scientists who discovered WZ-4-002, in order to advance the compound for clinical use as quickly as possible.

Third-party defendant Novartis Pharma, A.G. is a Swiss corporation with its principal place of business in Basel, Switzerland. It is one of the largest pharmaceutical companies in the world.

Third-party defendant Novartis International Pharmaceutical, Ltd. is a wholly-owned subsidiary of Novartis Pharma, A.G., and is a Bermuda company with its principal place of business in Bermuda. Novartis International Pharmaceutical, Ltd. is the original party to the Research Agreement with Dana-Farber which gave rise to the dispute at issue here.

Third-party defendant Novartis Institutes for Biomedical Research, Inc. is a wholly-owned subsidiary of Novartis International Phamaceutical, Ltd., incorporated in Massachusetts, with its principal place of business in Cambridge, Massachusetts. It is the assignee of the Research Agreement.

For simplicity's sake, I refer to the various Novartis third-party defendant entities collectively as "Novartis" in this Memorandum.

B. Non-Small-Cell Lung Cancer and Protein Kinase Inhibitors

Cancer can result from unchecked cellular growth and proliferation when protein kinases, such as epidermal growth factor receptor ("EGFR") and Jak3, are over-activated. Kinase inhibitors stunt such over-activation by binding to a specific protein kinase, effectively shutting off the biochemical reactions that cause cellular proliferation. By doing so, kinase inhibitors can be effective at treating certain cancers. Most kinase inhibitors bind only temporarily to the protein kinase, and the effect is temporary and reversible. If the patient stops taking the inhibitors, unchecked cellular growth may return. These are called "reversible inhibitors." Other inhibitors permanently bind to the protein kinase and are therefore called "irreversible inhibitors."

Two reversible protein kinase inhibitors, with commercial names Iressa and Tarceva, have been approved by the FDA to help fight non-small-cell lung cancer caused by the overactivation of the EGFR kinase. However, in some patients the cancer develops a resistance to Iressa and Tarceva. In 2004, it was discovered that the resistance develops largely because of a mutation in the EGFR kinase, called the T790M mutation. Until recently, Scientists searching for a way around this resistance had been unsuccessful. In 2008, Dana-Farber researchers discovered that an irreversible kinase inhibitor synthesized in 2007, WZ-4-002,

was effective at inhibiting the EGFR kinase with the T790M mutation.

C. Facts

1. The Dana-Farber-Novartis Research Agreement

On January 1, 2005, Dana-Farber and Novartis entered into the Research Agreement. Joint Appendix ("JA") Ex. 37 at 5. That Agreement provided that in exchange for funding from Novartis, Dana-Farber agreed to give Novartis first priority to license "Program Intellectual Property." *Id.* at 20 § 19.2. The Agreement defines Program Intellectual Property as "all proprietary rights in Program Technology . . . including patents, copyrights, mask works, trade secrets and proprietary information, and, in the case of a Principal Investigator[1] who receives Major Support,[·] any Invention conceived or reduced to practice by the Principal Investigator in his or her Field of

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[1] A Principal Investigator is defined in relevant part as "the Institute scientist in charge of a Funded Research Project under this Agreement and responsible for its conduct in accordance with the terms of the grant and the policies and procedures of the Institute." Joint Appendix Ex. 37 at 6 § 1. Each Principal Investigator is also offered a Consultancy Agreement with Novartis, in which, in exchange for a fee, the Investigator agrees not to consult with another commercial organization in a similar or overlapping area as the one the Investigator is working in for a Funded Research Project. *See id.* at 8 § 5.2(b) (offering a Consulting Agreement to Principal Investigators); *id.* at Ex. A-1 (sample Consulting Agreement).

[·] Major Support is defined as "direct cost funding (exclusive of overhead) to a single laboratory in an amount of [redacted] annually." *Id.* at 6 § 1. None of the scientists involved in this case received Major Support.

Endeavor[3] during a period in which he or she is receiving Major Support." *Id.* at 6 § 1.

Program Technology is defined as "Technology[4] invented, discovered or developed in whole or in part by an Institute Program participant," and "conceived or reduced to practice as part of a Funded Research Project . . . or by using the funds provided by Novartis for a Funded Research Project . . . ." *Id.* at 7 § 1.

A Funded Research Project is, in turn, defined as a research project "under the direction of an Institute scientist based upon a grant proposal that was submitted to and which was accepted by the Steering Committee . . . for funding pursuant to this Agreement." *Id.* at 6 § 1.

The Research Agreement also provided that "[t]o facilitate the Funded Research, the parties may choose to exchange compounds or materials that comprise one party's proprietary Technology," so long as researchers seeking to use Novartis proprietary Technology sign a Materials Transfer Agreement ("MTA"). *Id.* at 17 § 16. Dr. Michael Eck of Dana-Farber used a Novartis

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[3] A list of thirteen areas of research are defined as the fields of endeavor. "Kinase Oncogenes" is listed as one of them. *Id.* at 5 § 1.

[4] Technology is independently defined as "inventions, discoveries, innovations, knowledge, information and all other forms of technology whether or not protectable or protected by patent, copyright or trade secret law or otherwise." *Id.* at 7 § 1.

compound, [redacted] in his research pursuant to an MTA. In an application to use the Novartis compound, Dr. Eck stated that [redacted] will be used to study the sturyctre [sic] of their compoexes with JAK2 or JAK3 kinase, using x-ray crystallography." Under the MTA, Dr. Eck agreed that [redacted]

> will not be transferred or made available to any individual not under the supervision and control of [Dr. Eck] and will not be used for any purpose other than the Studies. This includes other Dana-Farber researchers who are not included in the collaboration with Novartis and other Dana Farber [sic] researchers who are included in the collaboration with Novartis, but who intend to use the Materials for research other than the Studies. JA Ex. 93 at 2.

Furthermore, Dr. Eck agreed that if

> the Materials are not solely used for the Studies, but for different studies which clearly are not encompassed under the above description of the Studies, or if the Materials are transferred to third parties . . . Novartis will own and the Applicants herewith assign all its rights to all patentable inventions, and know-how concerning the Materials which are discovered or improved during the course of such unauthorized studies. *Id.*

2. Funding Under the Research Agreement

Pursuant to the Research Agreement, Novartis has donated between [redacted] annually since 2005. *See* JA Ex. 37 at 7-8 § 5.1 (setting the 2005 funding amount at [redacted] and allowing for annual adjustments thereafter).

A Steering Committee, comprised of two Dana-Farber and two Novartis members, decides how to distribute the Novartis funding. *Id.* at 10 §§ 9.1-9.2. Each year, Novartis provides the Steering

Committee with Requests for Applications, to encourage Dana-Farber scientists to apply for grants funded under the terms of the Research Agreement. *Id.* at 11 § 10.1. Any grant applications are screened by a Pre-Clinical Grant Review Committee, which scores applications and gives suggestions to the Steering Committee as to which grant applications should be approved. *Id.* at 12 §§ 11.1-11.3. Once a grant application is approved, the Principal Investigator of the Funded Research Project is responsible for providing the Steering Committee with at least annual progress reports, "including a description of the work accomplished to date, a discussion and evaluation of the results of such work, and an assessment of progress made against goals established under the grant application or approval." *Id.* at 13 §§ 12.2-12.3.

3. Researchers and their Funding

I. Dr. Eck

Dr. Eck's specialty is using x-ray crystallography to determine the structure of kinases. Drs. Cai-hong Yun and Angela Toms are both post-doctoral fellows who worked under the direction of Dr. Eck.

a. 2005-2006 Funding and Reports

In 2004, Dr. Eck applied for funding under the Research Agreement for research projects to be conducted in 2005 and 2006. Among other things, his application sought [redacted] in funding to

help "provide a structural understanding of key molecular targets in oncology in order to facilitate inhibitor design," and focused on three different categories of proteins. One of those categories was the Jak2 and Jak3 family of kinases, which "are important targets for development of anti-leukemic and immunosuppressive drugs." Dr. Eck planned on studying the Jak2 and Jak3 kinases "with Novartis compounds in order to facilitate inhibitor optimization." Dr. Eck was listed as the Principal Investigator on his application.

When the funding was granted, Dr. Eck signed a Consulting Agreement with Novartis. From 2005 to 2006, Dr. Eck agreed "not to consult for another commercial organization in any area that is the same as or materially overlaps" with the research funded by Novartis. In 2005 and 2006, that area was "Epigenetic Drug Targets." The Novartis funding was also used in part to fund Drs. Yun[9] and Toms.

In his 2006 progress report, Dr. Eck stated that his team had "determined the structure of the Jak3 kinase domain in complex with Novartis inhibitor [redacted]." He also stated that

[9] While Dr. Yun researched EGFR at this time, that work was pursuant to a grant from the National Institute of Health through a grant application entitled "Inhibitor-sensitive-and-resistant EGFR mutants from lung cancer and glioblastoma."

they were "in the process of expressing the T790M resistance mutant of the EGFR for structural analysis with inhibitors."[6]

b. 2007-2008 Funding and Reports

Dr. Eck applied for further funding in 2006 for projects to be completed in 2007 and 2008. His application described the successes he had during the 2005-2006 grant period, which included providing "mission-critical resources to Novartis for the Jak project, including the first reported structure of a Jak-family kinase and structures of Novartis inhibitors in complex with Jak3." It stated that his aim with the new grant funding would be to "provide structural information to Novartis that is potentially invaluable for inhibitor development . . . . Additionally, we will undertake co-crystallization studies with Novartis inhibitors." The funding he received pursuant to the 2006 application in part paid the salary of Dr. Toms. Dr. Yun

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[6] Dr. Eck stated in his deposition that the T790M research was pursuant to his grant from the NIH, not Novartis's grant. Gatekeeper argues Dr. Eck's report to Novartis included the T790M research because Dana-Farber scientists were encouraged under the Agreement to share all of their research with Novartis, whether or not it was pursuant to a funded research project. *See* JA Ex. 37 § 28.2 ("To promote the mutual benefits of collaboration intended under the Program, the parties will encourage mutually productive and continuing interchanges between Institute scientists and Novartis scientists. Accordingly, the Institute and Novartis will use reasonable efforts to insure that Program Scientists are available to each other for consultation in the area of their respective projects and that they communicate with each other and exchange information regarding the Program and results of research projects, with the goal of enhancing the effectiveness of the collaboration.").

did not receive any funding pursuant to the 2006 application. Dr. Eck again signed a Consulting Agreement with Novartis with terms similar to those governing his 2005-2006 research, except that the area as to which he agreed not to consult was "Kinase Oncogenes."

In May 2007, Dr. Eck submitted a quarterly report to Novartis. Under the "Data Deliverables" section, Dr. Eck listed the "crystal structure of T790M mutant EGFR in complex with [redacted]" a specific kinase inhibitor. Under the "Hot Item" section, Dr. Eck listed the discovery that the "T790M EGFR mutation confers drug resistence by increasint [sic] the affinity for ATP, not by sterically interfering with inhibitor binding."

Dr. Eck also gave a presentation to Novartis in which he described his work on Jak3 using [redacted], and talked about the T790M mutation to the EGFR. When asked what he had presented regarding the T790M mutation, Dr. Eck said that he elaborated "on our sort of hot items, discovery of the sort of basis for resistance of the EGFR T790M mutation, and describ[ed] crystal structures of that mutant in complex with [redacted], as well as with irreversible inhibitory HKI-272."

Dr. Eck submitted an additional report in November 2007. In that report, Dr. Eck stated that his team had

> analyzed the structure and kinectics of the lung cancer resistance mutant T790M, both alone and in combination with the activating L858R mutation. We discovered that contrary to popular belief, resistance is due to

> increased affinity for ATP, rather than steric hindrance with inhibitor binding. We have determined the structure of the T790M mutant in complex with Novartis inhibitor [redacted], and are initiating HTS with the L858R/T790M mutant with collaborators at GNF [an independent non-profit foundation funded by Novartis] (Markus Warmuth and colleagues).[7]

In April of 2008, Dr. Eck submitted another quarterly report. He reported in a section titled Reagent Deliverables that "EGFR WT, L856R, and L858R/T790M kinase reagents" were deliverable to "GNF for assay development and HTS."

c. 2009-2010 Funding and Reports

In 2008, Dr. Eck applied for funding for research projects to be conducted in 2009 and 2010. The application's stated "long term goal is to understand at a structural level the catalytic regulation and receptor recognition of Jak-family kinases." The studies that would be performed with Novartis funding would "facilitate development of novel inhibitors specific for individual Jak kinases and may provide an avenue for discovery of allosteric regulators of Jak kinases including Jak2 and Jak3." The application again aimed to deliver "structural information to Novartis that is potentially invaluable for inhibitor development . . . ." Dr. Eck was again listed as the Principal Investigator, and Drs. Toms and Yun received funding from the grant. Dr. Eck

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[7] As noted above, it appears the discussion of T790M was a result of the Research Agreement's encouragement of scientists to share all their research with Novartis, because Novartis's 2007-2008 grant to Dr. Eck was to study Jak3 and the L858R and G719S mutants of EGFR, not T790M.

signed a Consulting Agreement with Novartis having terms similar to the one governing 2007-2008 in the area of "Kinase Oncogenes."

ii. Dr. Gray

In 2006, Dr. Nathanael Gray left the Novartis Institute for Functional Genomics ("GNF") to become a Principal Investigator at Dana-Farber and a member of the Harvard Medical School faculty. Dr. Gray's specialty was the "Bcr-abl" kinase.

Initially, Dr. Gray was given approximately $1.5 million by Dana-Farber as "start up" costs, until he could secure outside funding. In April 2006, Dr. Gray worked with Dr. Eck to do crystal structures of Bcr-abl. Afterwards, in October 2006, Dr. Gray collaborated with Dr. Eck to identify an irreversible inhibitor of the Jak3 kinase. Dr. Gray noted to Dr. Eck in January 2007 that Jak3 and EGFR, along with some other kinases, have a cysteine in their ATP binding cleft, which means that a similar strategy could be adopted for developing inhibitors that would target those kinases.

In June 2006, Dr. Gray applied for funding from Novartis for projects between 2007 and 2008. His application stated that his long term goal was "to develop chemical strategies for selectively inhibiting kinases bearing mutations to the gatekeeper residue," like the T790M mutation in EGFR. Dr. Gray's grant application was accepted and he signed a Consulting Agreement with Novartis. Pursuant to that agreement, from 2007-

2009 Dr. Gray agreed not to consult for another commercial organization in the area of "Kinase Oncogenes." Dr. Gray's post-doctoral research assistant, Dr. Wenjun Zhou, did not receive any of Dr. Gray's Novartis funding.

In 2007, Dr. Gray filed an annual progress report. He reported that he had not focused primarily on using "rational design to prepare selective inhibitors of 'gatekeeper' mutants of Bcr-abl, EGFR, c-Kit, and PDGFRbeta" as had been originally planned, but that he would place more emphasis on that in 2008. His team had, however, "synthesized approximately 150 new type II inhibitors covering approximately 10 distinct scaffold classes. . . . A couple of scaffolds also inhibit the gatekeeper mutant of EGFR (T790M) showing that the approach is general."[8] In describing the significance of this development, Dr. Gray stated that the compounds his team had developed "will serve as good starting points for optimization for numerous kinase projects. This may allow medicinal chemistry projects to start with scaffolds that will be suitable to address resistance mutants."

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[8] Dr. Gray later stated in his deposition that the scaffolds referenced with respect to the T790M mutant were also Type 2 inhibitors (which exploit an additional binding site immediately adjacent to the region occupied by ATP in the kinase), instead of Type I inhibitors (which target the ATP binding site of the kinase itself).

iii. Dr. Jänne

Dr. Pasi Jänne is a Dana-Farber researcher who has specialized in the EGFR kinase and its relationship to non-small-cell lung cancer since at least 2004. In 2004 and 2005, Dr. Jänne and two other Dana-Farber researchers discovered and published papers on the T790M mutation of the EGFR which causes resistance to the two existing EGFR kinase inhibitors, Iressa and Tarceva. Dr. Jänne and his research assistant have never received funding from Novartis, instead funding their research through grants from the National Institute of Health.

4. Discovery of WZ-4-002

In 2004, Dr. Jänne discovered the T790M mutation of the EGFR kinase, and began publishing papers on it starting in 2005. Dr. Eck was "immediately interested" in Dr. Jänne's discovery, and in 2005, had his research assistant Dr. Yun begin studying the T790M mutation. At the time, Dr. Yun was being funded in part by a grant under the Research Agreement to study the Jak2 and Jak3 kinases, but was also being funded by a NIH grant specific to EGFR mutants, such as T790M.

In 2005, Dr. Eck was also performing co-crystallization studies of Jak3 and [redacted] the Novartis-patented inhibitor. Dr. Eck provided the results of these studies to Dr. Gray. Dr. Eck suggested to Dr. Gray that they try to make irreversible

inhibitors based on the findings of the co-crystallization studies.

In 2006 and 2007, Drs. Gray and Zhou created a library of irreversible kinase inhibitors, including one, WZ-1-84, which was an irreversible form of Novartis [redacted]. When testing whether WZ-1-84 was capable of irreversibly inhibiting Jak3, Drs. Gray and Toms (who was also funded by a grant under the Research Agreement) found that the compound demonstrated no cellular activity. To investigate why, in February 2007 they prepared a crystal structure of WZ-1-84 with the T790M mutation of the EGFR, because it "is somewhat similar to JAK3" and they thought that it "should be informative." As a part of his project to create a library of irreversible inhibitors, Dr. Zhou also created WZ-4-002. According to his notebook, Dr. Zhou first synthesized WZ-4-002 on May 31, 2007.

Around the same time, Dr. Eck discovered that the T790M mutation resisted existing treatments, not by a structural change, but by increasing the mutated kinase's affinity for ATP. This allows ATP to out-compete the existing inhibitors for the EGFR kinase binding site, preventing the inhibitors from effectively checking cellular growth. Because there were no physical mutations to the kinase, Drs. Jänne and Eck proposed that they, along with Dr. Gray, should attempt to screen T790M inhibitors directly against the mutant form of the kinase, rather

than first testing the inhibitors against the "normal" kinase and then adjusting them for physical alterations in the mutation. Dr. Gray initially resisted the idea, and, as a result, Dr. Eck went to GNF, an independent Novartis-funded non-profit, to obtain EGFR with the T790M mutation on which to test irreversible kinase inhibitors.

Dr. Gray continued testing his library of inhibitors against various kinases, including EGFR (but not the T790M mutation) in 2008. In the summer of 2008, Dr. Jänne approached Dr. Gray and asked if he could use Dr. Gray's library of inhibitors to see if any were effective against the EGFR T790M mutation. Dr. Zhou gave a series of inhibitor compounds from the library he had created to Dr. Jänne's research assistant. Though Drs. Gray and Zhou did not perform the tests themselves, Dr. Gray remained in touch with Dr. Jänne regarding the progress of the tests. When Dr. Jänne reported early success with some of Dr. Gray's irreversible inhibitors, Dr. Gray suggested the order in which Dr. Jänne should test the remaining inhibitors to maximize testing efficiency with the T790M mutation.

One of the earliest compounds Dr. Jänne tested was WZ-4-002, which, he discovered, effectively inhibited the EGFR kinase with the T790M mutation. Drs. Gray and Zhou then began selecting compounds they thought would be equally likely to be effective, and sent them to Drs. Eck and Yun for x-ray crystallography.

In October 2008, Drs. Gray, Zhou, Jänne, and Eck filed a Dana-Farber Invention Disclosure Form disclosing the effectiveness of the WZ-4-002 compound on the T790M mutation of the EGFR kinase. In the section disclosing the sources of funding that supported the work leading to the invention, only two grants were listed, neither of which was from Novartis. Drs. Jänne and Gray signed the form, assigning their rights to the invention to Dana-Farber. Though Dr. Eck had made suggestions and edits for the form, none of his edits appear to have made it into the final version signed by Drs. Jänne and Gray.

5. The '419 Patent and Nature *Article*

On May 5, 2009, Drs. Gray, Eck, and Jänne submitted patent application number 61/215,419 for WZ-4-002 (the "'419 patent"). The application claimed "novel pyrimidine, pyrrolo-pyrimidine, purine and triazine compounds which are able to modulate epidermal growth factor receptor (EGFR), including Her-kinases, and the use of such compounds in the treatment of various diseases, disorders or conditions." Some of the claims in the patent, however, are broader. In particular claim 53 claims a method of inhibiting a cysteine residue kinase "wherein the cystine residue is located in or near the position equivalent to Cys 797 in EGFR, including Jak3, Blk, Bmx, Btk, HER2 (ErbB2), HER4 (ErbB4), Itk, Tec, and Txk."

In December 2009, Drs. Zhou, Yun, Eck, Gray, Jänne and others published a paper in *Nature* titled "Novel mutant-selective EGFR kinase inhibitors against EGFR T790M." That paper disclosed much of the research done to discover that WZ-4-002 could effectively inhibit the T790M mutation of the EGFR kinase. Although the paper listed a number of sources of funding for the project, it did not list Novartis as having provided any funding.

6. Gatekeeper and the Patent License

Two months before submitting the patent application, on March 18, 2009, Drs. Gray and Jänne founded Gatekeeper with Jeff Engelman, Kwok-Kin Wong, and John Chant, and began negotiations with Dana-Farber for license rights to WZ-4-002. Dr. Eck did not join Gatekeeper in part because the other founders were concerned about a conflict between his Consulting Agreement with Novartis in the area of kinase oncogenes.

Then, on June 1, 2009, Dana-Farber entered into an option agreement with Gatekeeper. Gatekeeper could exercise its option for a license to the '419 patent within twelve months provided that it obtained at least $250,000 in financing. If Gatekeeper exercised its option within twelve months, Gatekeeper and Dana-Farber would have 180 days to negotiate and execute a license agreement.

Dana-Farber conducted an internal inquiry to determine whether the WZ-4-002 discovery fell under the Research Agreement

with Novartis such that Novartis might have first priority to a license. On the basis of representations made by Dr. Gray regarding the funding involved in the WZ-4-002 discovery, Dana-Farber determined that the WZ-4-002 discovery did not implicate the Research Agreement.

In December 2009, around the same time as the publication of the paper in *Nature*, Novartis learned of WZ-4-002's discovery, and made a claim to Dana-Farber that it was covered under the Research Agreement. Novartis claimed that WZ-4-002 was "Program Intellectual Property" pursuant to a "Funded Research Project" by Dr. Gray under the Novartis Agreement, and that Novartis therefore had first priority for licensing the '419 patent.

On April 23, 2010, Gatekeeper gave Dana-Farber notice that it had raised the necessary funds pursuant to the option agreement and was exercising its option for the '419 patent. Dana-Farber informed Gatekeeper that Novartis was claiming priority under the Research Agreement. Faced with conflicting claims of priority, Dana-Farber investigated Dr. Gray's claim that WZ-4-002 was not discovered using Novartis funds or grants.

In August 2010, Dana-Farber concluded that WZ-4-002 was subject to the Research Agreement, and notified Gatekeeper.[4]

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[4] Citing attorney-client privilege, Dana-Farber has not released its analysis of why it thought WZ-4-002 constituted Program Intellectual Property under the Research Agreement.

When Gatekeeper notified its financial backers, they withdrew their funding.

D. Procedural History

On September 21, 2010, Dana-Farber filed the instant complaint for a declaratory judgment to determine the rights and responsibilities of Gatekeeper and Dana-Farber with regard to WZ-4-002. Gatekeeper filed an answer and third-party complaint against Novartis. Numerous counter- and cross-claims were filed against various Novartis entities.

During the course of the proceedings, Novartis determined that it was not interested in pursuing the '419 patent license. Thus, at a hearing on August 19, 2011, I recognized that the issues remaining go only to entitlement and damages: (1) whether Gatekeeper was entitled to a license under the option contract, (2) whether Novartis had priority over Gatekeeper under the Research Agreement, and (3) if Gatekeeper was entitled to the license, what damages Gatekeeper incurred as a result of the delay in obtaining the license.

On December 1, 2011, Novartis filed a motion for summary judgment on Gatekeeper's claim for intentional interference with contractual relations. Gatekeeper filed a cross-motion for summary judgment.

II. STANDARD OF REVIEW

A movant is entitled to summary judgment when "the movant shows that there is no genuine dispute as to any material fact and the movant is entitled to judgment as a matter of law." Fed. R. Civ. P. 56(a). "A dispute is genuine if the evidence about the fact is such that a reasonable jury could resolve the point in the favor of the non-moving party," and "[a] fact is material if it has the potential of determining the outcome of the litigation." *Farmers Ins. Exch.* v. *RNK, Inc.*, 632 F.3d 777, 782 (1st Cir. 2011) (citation omitted). "[C]onclusory allegations, improbable inferences, and unsupported speculation" are insufficient to create a genuine dispute of material fact to defeat a motion for summary judgment. *Sullivan* v. *City of Springfield*, 561 F.3d 7, 14 (1st Cir. 2009) (quotation and citation omitted).

As I must, I "view the facts in the light most favorable to the party opposing summary judgment." *Rivera-Colón* v. *Mills*, 635 F.3d 9, 10 (1st Cir. 2011). Because I am addressing cross-motions for summary judgment, I "must view each motion, separately, through this prism." *Estate of Hevia* v. *Portrio Corp.*, 602 F.3d 34, 40 (1st Cir. 2010).

III. DISCUSSION

To support a claim of intentional interference with contractual relations under Massachusetts law, a party must show "(1) the existence of a contract or a business relationship which contemplated economic benefit; (2) the [other party's] knowledge of the contract or business relationship; (3) the [other party's] intentional interference with the contract or business relationship for an improper purpose or by improper means; and (4) damages." *Howcroft* v. *City of Peabody*, 747 N.E.2d 729, 748-49 (Mass. App. Ct. 2001) (quoting *Swanset Dev. Corp.* v. *Taunton*, 668 N.E.2d 333, 338 (Mass. 1996)).

The parties cross-move for summary judgment with respect to entitlement to license the '416 patent, the basis for the third element of Gatekeeper's tortious interference with contract claim. The '416 patent contains many distinct claims, but only those that relate to the use of WZ-4-002 as an effective inhibitor of the T790M mutation of the EGFR kinase are relevant to this action. Each claim must be treated as its own invention, *Jones* v. *Hardy*, 727 F.2d 1524, 1528 (Fed. Cir. 1984), and Gatekeeper's cross-complaint alleges that Novartis improperly asserted entitlement to license the '416 patent to develop WZ-4-002 as an effective inhibitor of the T790M mutation of the EGFR kinase. Therefore, the relevant inquiry is not whether Novartis can properly assert entitlement with respect to any

claim in the '416 patent, but whether it can properly assert entitlement with respect to the claims for which Gatekeeper alleges tortious interference.

A. Novartis's Motion for Summary Judgment

Novartis has moved for summary judgment on the grounds that (1) Novartis was entitled to license the '419 patent pursuant to the Research Agreement; (2) Gatekeeper was not entitled to the option to license the '419 patent because Dr. Eck's and Dr. Gray's consulting agreements prevented them from consulting or collaborating with Gatekeeper in the field of kinase oncogenes; and (3) Novartis was additionally entitled to license the '419 patent pursuant to a Materials Transfer Agreement with Dr. Eck. If Novartis is correct on any of these grounds, Gatekeeper cannot support its claim of intentional interference with contractual relations. For purposes of Novartis's motion for summary judgment, I view the facts in the light most favorable to Gatekeeper.

1. Entitlement Under the Research Agreement

Under the Research Agreement, Novartis could claim licensing priority over the '419 patent if it was "invented, discovered or developed in whole or in part by an Institute Program participant," and was "conceived or reduced to practice as part of a" research project "under the direction of an Institute scientist based upon a grant proposal that was submitted to and

which was accepted by the Steering Committee . . . for funding pursuant to [the Research] Agreement." Thus, Novartis must show that: (1) the '419 patent was developed at least in part by an Institute Program Participant, defined to include any Dana-Farber scientists "engaged in scientific activities under [the Research Agreement];" (2) it was conceived or reduced to practice; and (3) it was done so using grant funds obtained from Novartis under the Research Agreement.

The first element is not in dispute to the extent that the parties recognize that Dana-Farber scientists "engaged in scientific activities" under the Research Agreement ("Institute Program participants") were involved in the discovery of all of the compounds here. However, there is some disagreement over the meaning of "in part." Novartis argues that "in part" is quite broad, encompassing ideas that are passed between researchers, information that they share, and sources of inspiration for further independent work. It claims that elements of the '419 patent were discovered "in part" using funding from Novartis because (a) the "original seed" of applying irreversible inhibitors to kinases to inhibit cellular proliferation came from Dr. Eck's Novartis-funded work with Jak3; (b) Dr. Eck discovered that the T790M mutant of the EGFR kinase worked by increasing the kinase's affinity for ATP rather than changing its shape; and (c) it was only after Dr. Eck shared his ideas with Drs. Gray and

Zhou that they began working on WZ-4-002. Gatekeeper, on the other hand, mostly ignores Novartis's implied definition of "in part" and focuses solely on the direct line of steps from Dr. Zhou's creation of WZ-4-002 to Dr. Jänne's discovery that WZ-4-002 inhibits the T790M mutant. As will become apparent below, I am of the view that Novartis's implied understanding is too broad, and that Gatekeeper's (apparent) assumption more accurately captures the meaning of "in part."

i. Conceived or Reduced to Practice

To say that an invention must be "conceived or reduced to practice" is to invoke terms of art in patent law. In interpreting a contract, "[w]hen a term becomes, within a certain industry, a term of art, courts are to apply the technical meaning of the term instead of any other 'plain meaning' in general society." *In re Settlement Facility Dow Corning Trust*, 628 F.3d 769, 776 (6th Cir. 2010).

As a term of art, "conception" requires that the inventor have a specific, settled idea such that "one skilled in the art could understand the invention." *Burroughs Wellcome Co.* v. *Barr Labs., Inc.*, 40 F.3d 1223, 1228 (Fed. Cir. 1994). The inventor must be able to "describe his invention with particularity," *id.*, because with chemical compounds "[c]onception does not occur unless one has a mental picture of the structure of the chemical, or is able to define it by its method of preparation, its

physical or chemical properties, or whatever characteristics sufficiently distinguish it." *Amgen, Inc.* v. *Chugai Pharm. Co.*, 927 F.2d 1200, 1206 (Fed. Cir. 1991).

Conception also requires "a definite and permanent idea of the complete and operative invention, as it is hereafter to be applied in practice." *Id.* (quotation omitted) (emphasis added). Therefore, simple synthesis and understanding of the structure of a compound does not satisfy the requirements of conception in the absence of an understanding of either applications in practice, or experimentation to determine the compound's chemical properties.

Given the difficulty of describing a chemical compound with sufficient particularity for patentability until it has been tested through experimentation, conception and reduction to practice generally occur simultaneously for chemical compounds. *See* Chisum on Patents § 10.04(5). To show that an idea has been reduced to practice, "the inventor must prove that he constructed an embodiment or performed a process that met all the limitations of the claim, and that he determined that the invention would work for its intended purpose." *Slip Track Sys., Inc.* v. *Metal-Lite, Inc.*, 304 F.3d 1256, 1265 (Fed. Cir. 2002). "The adequacy of a reduction to practice is to be tested by what one of ordinary skill in the art would conclude from the results of the

tests." *Id.* (quoting *Winter* v. *Lebourg*, 394 F.2d 575, 581 (C.C.P.A. 1968)).

Here, although WZ-4-002 was first synthesized on May 31, 2007, it was neither conceived nor reduced to practice on that date because its inventor had not discovered its effect on the T790M mutation of the EGFR kinase. Instead, it was only truly conceived and reduced to practice when it was found to be effective at inhibiting the EGFR kinase with the T790M mutation during Dr. Jänne's, Dr. Gray's, and Dr. Eck's research in the summer of 2008.[10] It is undisputed that Dr. Jänne's lab was the

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[10] Novartis claims that the reduction to practice occurred no later than February 2007, when Dr. Gray found that some of the compounds claimed in the '419 patent were effective kinase inhibitors. For example, WZ-1-84 was found to have cellular activity on Jak3 and Bmx kinases in February 2007. This, Novartis argues, was sufficient to show reduction to practice for the entirety of the '419 patent claims, because a "prior reduction to practice of the species precludes another party from claiming that he is the first inventor of the genus containing the species." *Mikus* v. *Wachtel*, 504 F.2d 1150, 1151 (C.C.P.A. 1974). This argument proves too much. The discovery that an irreversible kinase-inhibiting compound was effective against Jak3 and Bmx does not mean it will be effective against the T790M mutation of EGFR. Indeed, the T790M mutation posed a particular challenge precisely because existing kinase inhibitors were ineffective against it. And, to use the inapt analogy from the case Novartis cites, one compound being effective against one type of kinase does not mean it is the species to another compound's genus especially when it is effective against a different type of kinase (albeit one that is structurally similar). As Novartis itself notes, patent law treats each claim as a separate invention. Thus, even assuming (without deciding) that some of the '419 patent's claims were reduced to practice in February 2007, that does not mean that WZ-4-002's effective reduction to practice date was also February 2007.

source of the discovery, through testing, that WZ-4-002 was effective as applied to the T790M mutation.[11]

ii. Sources of Funding for WZ-4-002

Under the terms of the Research Agreement, Novartis is entitled to a license of WZ-4-002 only if it was "conceived or reduced to practice as part of a Funded Research Project," that is, a research project "based upon a grant proposal that was submitted to and which was accepted by the Steering Committee." Since WZ-4-002 was only created on May 31, 2007, and it was not until the summer of 2008 that it was discovered to be an effective inhibitor of the T790M mutation of EGFR kinase, Novartis must show that the scientists involved were working on a research project funded by Novartis in the summer of 2008.[12]

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[11] Novartis notes that the '419 patent contains 92 claims, not all of which relate exclusively to the T790M mutation of the EGFR kinase. *See, e.g.*, JA Ex. 11 at claim 53 (claiming a method of inhibiting a cysteine residue kinase "wherein the cysteine residue is located in or near the position equivalent to Cys 797 in EGFR, including Jak3, Blk, Bmx, Btk, HER2 (ErbB2), HER4 (ErbB4), Itk, Tec, and Txk"). However, as noted above, what matters for purposes of this case is the conception and reduction to practice of WZ-4-002, because each claim of the '419 patent is a separate invention.

[12] Novartis's main argument in its briefs is that the creation of WZ-4-002 and the idea of applying irreversible inhibitors to the T790M mutation was inspired by or else relied on information from pre-2007, Novartis-funded research projects in Dr. Eck's and Dr. Gray's labs. However, this argument fails, because inspiration and the provision of ideas is not equivalent to "conceived or reduced to practice." Indeed, nearly all scientific work is inspired by or based on information or ideas from other scientists. It cannot be that Novartis is entitled to

As noted above, only the labs of Drs. Eck, Gray, and Jänne were involved in the discovery of the effectiveness of WZ-4-002 against the T790M mutation. Dr. Jänne has never received funding under the Novartis Research Agreement. Nor has Dr. Zhou. Thus, their contributions to the discovery of WZ-4-002 cannot serve as the basis for Novartis's claim.

To show that Novartis funds were used to conceive WZ-4-002 or reduce it to practice as an inhibitor of the EGFR kinase T790M mutation, Novartis strings together a number of different projects over a four-year period in an effort to create a chain of Novartis-funded researchers. Its claim rests upon the following sequence of events: in 2005 and 2006, Dr. Yun made copies of the T790M mutation.[13] Around the same time, Dr. Eck performed the co-crystallization studies of Jak3 and [redacted] which gave him the idea to have Dr. Gray's lab make irreversible inhibitors based off a similar structure to [redacted]. Dr. Toms also studied Jak3 and [redacted] and performed a structural modeling exercise with them. This is said to have given Dr. Zhou the idea how to design his library of irreversible inhibitors,

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a license to every invention ever made which is inspired by a discovery made during a Novartis-funded research project.

[13] As noted above, however, Dr. Yun was operating under a NIH grant at this time supporting his research into EGFR. *See supra*, Section I(C)(3)(i)(a); Section I(C)(4). The Novartis grant supporting Dr. Yun in 2005-2006 was for research into the Jak3 kinase, not EGFR.

including WZ-4-002, which fell within Dr. Gray's Funded Research to target gatekeeper mutations of EGFR.

However, the relevant time period for the discovery of WZ-4-002's effectiveness against the T790M mutation is somewhere between May 31, 2007 and the end of 2008. In January 2007, Dr. Eck discovered that the T790M mutation was not a physical mutation of the kinase, but a preference-based mutation which merely increased the EGFR kinase's affinity for ATP. He therefore suggested that Drs. Jänne and Gray screen inhibitors directly against T790M. Dr. Gray refused, so Dr. Jänne and Dr. Eck did it themselves. Dr. Eck obtained the requisite T790M mutated EGFR from GNF, not from any T790M mutations created by Dana-Farber, in late August or early September 2008. Dr. Jänne borrowed Dr. Zhou's library of inhibitors to test against T790M, including WZ-4-002 (which was created at the end of May 2007), and discovered the compound's effectiveness in either September or October 2008. It was then that WZ-4-002 was reduced to practice, and therefore it is within this time period that I must look to see whether the reduction to practice occurred under a Funded Research Project.

The only Novartis grants during this time period provided to scientists arguably involved in the reduction of WZ-4-002 to practice are (1) Dr. Gray's grant for 2007-2008 entitled "Design of Inhibitors of Drug-Resistant Protein Kinases," JA Ex. 2,

(2) Dr. Eck's grant for 2007-2008 entitled "Crystallographic analysis of Jak2 and PI3-K and their inhibitor complexes," JA Ex. 85, and (3) Dr. Eck's 2009-2010 grant entitled "Structural and Mechanistic studies of Jak2 and drug-resistant EGFR mutants," JA Ex. 114. None of these grants cover WZ-4-002.

a. Dr. Gray's 2007-2008 Grant

Dr. Gray's grant application expressed three aims, only the first of which is relevant here. That aim was to:

> Use rational design to prepare selective inhibitors of "gatekeeper" mutants of Bcr-Abl, EGFR, c-Kit, and PDGFRb. We will use a recently developed pharmacophore model for inhibitors that bind to the "inactive" or "DFG-loop out" kinase conformation to prepare new kinase inhibitors and use a broad cellular kinase-selectivity panel to assess their selectivity and utility. Compounds will be synthesized by a combination of solution and solid-phase approaches, purified by reverse-phase HPLC and subject to cellular and biochemical profiling in collaboration with the kinase platform at the Genomic Institute of the Novartis Research Foundation (GNF). Deliverable is new potential chemical composition of matter and associated biological profiling data.

The effectiveness of WZ-4-002 with the T790M mutation was due to its covalent bond, which made it irreversible, but Dr. Gray's grant application does not mention an irreversible covalent bond targeting the T790M mutation at all. Instead, the grant application discloses that Dr. Gray was going to use a pharmacophore model binding to the inactive kinase conformation; that is, instead of binding to the gateway, as WZ-4-002 does, Dr. Gray was intending to make "new ATP-site targeting inhibitors

with optimized interactions in the hinge-region binding area and 'allosteric pocket' that are capable of accommodating various mutations to the gatekeeper position." This is an entirely different method of attacking the kinase, and therefore Dr. Gray's grant application cannot be the source of funding under which WZ-4-002 was reduced to practice.

b. Dr. Eck's 2007-2008 Grant

Dr. Eck's 2007-2008 grant, entitled "Crystallographic Analysis of Jak2 and PI3-K and their Inhibitor Complexes" also does not cover WZ-4-002. In that grant application, Dr. Eck noted that his team was "studying two well-validated kinase targets in oncology, the tyrosine kinase Jak2 and lipid kinase PI3kα." The goal of the grant application was "to determine the three-dimensional structures of these proteins in their intact, regulated form in order to understand their respective regulatory mechanisms and to understand how oncogenic mutations disrupt these interactions to cause cancer." Thus, the specific aims of the grant applications were to (1) "express, purify, crystallize and determine the three-dimensional structure of full-length Jak2," (2) "take a domain-based approach to dissecting the regulatory mechanism and receptor recognition of Jak2," and (3) perform "[c]rystallographic studies of PI3Kα."

None of these aims or goals involve EGFR, or the T790M mutation, and none are related at all to creating a compound to

form an irreversible covalent bond to inhibit the over-expression of the EGFR kinase. Thus, this grant, too, cannot have been the funding under which WZ-4-002 was reduced to practice.

c. Dr. Eck's 2009-2010 Grant

Finally, Dr. Eck's 2009-2010 grant application, entitled "Structural and mechanistic studies of Jak2 and drug-resistant EGFR kinase mutants" could not have been the funding used for the discovery of WZ-4-002's effectiveness, because funding did not begin until January 1, 2009. Dr. Jänne discovered WZ-4-002 to be effective against the T790M mutation in the summer of 2008.

Furthermore, that grant application was not on point. Specifically, the grant application's goal with respect to EGFR was to:

> facilitate development of drugs to treat the drug resistant EGFR mutants in lung and other cancers. In particular, we focus on the L858R/T790M and exon20 InsNPG mutants. Our Specific Aim 2 is to determine the structures of these drug resistant mutants in complex with inhibitors and to characterize their kinetic and drug binding properties. We will work closely with Novartis and GNF colleagues to discover and characterize *reversible* small molecule inhibitors that exhibit specificity for the L858R/T790M mutant versus the WT EGFR.

(emphasis added). The focus on reversible inhibitors was, in Dr. Eck's opinion, because

> [a]lthough the L858R/T790M mutant is efficiently inhibited by irreversible inhibitors such as EKB-569 and HKI-272[36, 37, 41-43], we believe that current irreversible inhibitors are very unlikely to succeed in the clinic. Because the dose-limiting toxicities of

> EGFR inhibitors are likely caused by "on-target" inhibition of WT EGFR (skin rash and diarrhea) and the irreversible inhibitors lack specificity for the mutant kinase, we reason that there can be little if any "therapeutic window" for their effective use. Potential liabilities arising from their reactive Michael-acceptor groups may also limit their clinical development. As demonstrated by the serendipitous specificity of gefitinib and erlotinib for the L858R and deletion mutants, the obvious solution is to develop next-generation compounds that inhibit the L858R/T790M mutant more potently that [sic] WT EGFR.

(emphasis in original).

Thus, Dr. Eck's 2009-2010 grant application could not have been the source of funding for the discovery of WZ-4-002's efficacy against T790M, because (a) the grant began funding Dr. Eck after WZ-4-002 had been found to be effective against the T790M mutation, and (b) the grant application sought to discover reversible, not irreversible, inhibitors of the T790M mutant.

Therefore, Novartis cannot show under the Research Agreement that it was the source of funding for the discovery of WZ-4-002's efficacy in inhibiting the T790M mutant of the EGFR kinase.

2. Gatekeeper Could Not Obtain a License

Next, Novartis argues that under the Consulting Agreement with Dr. Gray and the Research Agreement with Dana-Farber, Gatekeeper could not obtain a license to WZ-4-002.

i. Dr. Gray's Consulting Agreement

In Dr. Gray's Consulting Agreement, he agreed "not to consult for another commercial organization in any area that is

the same as or materially overlaps with any of the Funded Research Projects currently funded by [Novartis] under the Program, as determined by the Steering Committee" in the field of "Kinase Oncogenes." However, Dr. Gray and Novartis simultaneously signed the Dana-Farber Standard Consulting Agreement Provisions, which were attached to the Consulting Agreement between Dr. Gray and Novartis. Those Provisions, which Dr. Gray and Novartis agreed "shall govern" over any conflict with the Consulting Agreement, provided that Novartis:

> agrees and understands that [Dr. Gray] has a pre-existing obligation to assign to his or her employer, [Dana-Farber], all of [Dr. Gray]'s rights in intellectual property which arise or are derived from [Dr. Gray]'s employment at [Dana-Farber] or which utilize the funds, including funding from any outside source awarded to or administered by [Dana-Farber], personnel, facilities, materials, or other resources of [Dana-Farber] including resources provided in-kind by outside-sources. [Novartis] has no rights by reason of this Agreement in any publication, invention, discovery, improvement or other intellectual property, whether or not publishable, patentable or copyrightable that is subject to [Dr. Gray]'s obligations to [Dana-Farber]. [Novartis] also acknowledges and agrees that it will enjoy no priority or advantage as a result of the consultancy created hereunder in gaining access, whether by license or otherwise, to any proprietary information or intellectual property of [Dana-Farber].

The express terms adopted by the parties pursuant to the Dana-Farber standard provisions state that Novartis has no priority to IP as a result of its consulting agreements with individual researchers. Therefore, the Consulting Agreement

between Dr. Gray and Novartis does not entitle Novartis to WZ-4-002.

Novartis, however, argues that the Consulting Agreement prevents Gatekeeper from obtaining a license, because Gatekeeper could not be a "bona fide purchaser" of the '419 patent. Section 1 of the Consulting Agreement provides that if Dr. Gray "desires to establish a consulting agreement with another pharmaceutical firm at the same time as this agreement is in effect, [Dr. Gray] will inform the Steering Committee about such an intention and obtain prior written approval from the Steering Committee to enter into such an arrangement." Neither party has provided evidence of the Steering Committee's written consent for Dr. Gray to form Gatekeeper in the record before me.[14]

Instead, Gatekeeper argues that founding a company does not mean that Dr. Gray established a "consulting agreement" with it. Neither the Research Agreement nor Dr. Gray's Consulting Agreement define a "consulting agreement." Webster's New International Dictionary defines "consulting" when used as an adjective as "that advises: that aids esp. by providing professional or expert advice." *Webster's New Int'l Dictionary* 490 (3d ed. 1986). Agreements are "arrangements (as between two or more parties) as to a course of action." *Id.* at 43. Thus, as

[14] Gatekeeper claims Dr. Gray "sought and obtained" the Steering Commitee's consent, but there is no evidence of it in writing, as required by the Consulting Agreement.

relevant here, a consulting agreement is an arrangement for one party to aid another by providing professional or expert advice.

There is no evidence in the record that Dr. Gray had such an agreement with Gatekeeper.[15] Because Novartis bore the burden of proving Dr. Gray had violated his Consulting Agreement, its failure to provide proof that Dr. Gray's founding of Gatekeeper involved an agreement to aid Gatekeeper by providing expert advice is fatal to this line of Novartis's argument.

ii. Dana-Farber's Research Agreement

Next, Novartis argues that the Research Agreement prohibited Dana-Farber from entering into the Option Agreement with Gatekeeper, and therefore Gatekeeper was not entitled to a license for WZ-4-002. It cites section 14.2 of the Research Agreement, entitled "Research Support from Other Companies," for support. *See* JA Ex. 37 § 14.2.

Section 14.2 states that Dana-Farber "may enter into research support and collaboration agreements with third party commercial organizations . . . for cancer research in the laboratory of a Principal Investigator" as long as Novartis first had the opportunity to fund the research support or collaboration

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[15] Dr. Gray admitted to founding Gatekeeper with the purpose of developing applications of WZ-4-002, but that does not necessarily mean he intended to consult with it after its founding. The record is devoid of evidence that Dr. Gray is a consultant for Gatekeeper in addition to an investor in it.

if it was within any of the Fields of Endeavor, among other conditions. *Id.* The Research Agreement does not define "research support and collaboration agreements," so I again look to the plain meaning of those terms to determine whether the Option Agreement is subject to the restrictions contained in section 14.2.

Webster's New International Dictionary defines research as "studious inquiry or examination; esp: critical and exhaustive investigation or experimentation having for its aim the discovery of new facts and their correct interpretation, the revision of accepted conclusions, theories, or laws in the light of newly discovered facts, or the practical applications of such new or revised conclusions, theories, or laws." *Webster's New Int'l Dictionary* 1930 (3d ed. 1986). Support, when it is used as a noun, means "the act, process, or operation of supporting," that is, the act, process, or operation of "uphold[ing] by aid, countenance, or adherence." *Id.* at 2297. Collaboration is defined as "the act of collaborating," that is, the act of "work[ing] jointly esp. with one or a limited number of others in a project involving composition or research to be jointly accredited." *Id.* at 443. Finally, as discussed above, agreements are "arrangements (as between two or more parties) as to a course of action." *Id.* at 43.

Under the plain meaning of the terms, then, the Option Agreement was not a "research support or collaboration agreement[]." The Option Agreement provided Gatekeeper with an option for an exclusive license to develop WZ-4-002 commercially. An agreement for an option to a license to intellectual property is not an offer to research that intellectual property or to collaborate with the licensee on it. Thus, Novartis's argument that section 14.2 of the Research Agreement prevented Dana-Farber from entering into the Option Agreement with Gatekeeper fails. I note that Novartis appears to abandon this theory in its opposition to Gatekeeper's motion for Summary Judgment.

3. Entitlement Under the Materials Transfer Agreement

Under the Materials Transfer Agreement (the "MTA"), Dr. Eck agreed that in the event that [redacted] was "used for any purpose other than the" studies that Dr. Eck disclosed in his application (in this case, [redacted] was to be "used to study the sturyctre [sic] of their compoexes with JAK2 or JAK3 kinase, using x-ray crystallography"), then Dr. Eck and Dana-Farber agreed that Novartis would own or have a license to "all patentable inventions, and know-how concerning the Materials which are discovered or improved during the course of such unauthorized studies."

Novartis argues that Dr. Eck violated the MTA by providing Dr. Gray with information about Dr. Eck's studies of [redacted] and

its structure. This is an overreading of the MTA. The MTA is most logically read as prohibiting Dr. Eck from transferring the physical samples of [redacted] he was given, not information he gleaned as a result of using them. Specifically, the MTA's prohibition is as follows:

> The Material will not be *transferred* or made available to any individual not under the supervision and control of Investigator and will not be *used* for any purpose other than the Studies. This includes other Dana-Farber researchers who are not included in the collaboration with Novartis and other Dana Farber researchers who are included in the collaboration with Novartis, but who intend *to use* the Materials for research other than the Studies. Upon completion of the Studies, *any unused Material will be destroyed* under the Applicants' supervision in accordance with the applicable laws and regulations, and the instructions of Novartis, if any.

(emphasis added). As the above-emphasized words taken in context make clear, the MTA's prohibition is on the unauthorized transfer or use of the physical sample of [redacted] that Dr. Eck borrowed from Novartis pursuant to the MTA. The MTA's direction that "unused" Material be "destroyed" would otherwise not make sense.

Novartis does not argue that Dr. Eck transferred any of the samples of [redacted] to Dr. Zhou. Dr. Zhou and Dr. Gray both stated that no sample of [redacted] ever passed between their lab and Dr. Eck's. There is no evidence that it ever did so. Thus, Dr. Eck did not violate the plain terms of the MTA and Novartis cannot claim priority over WZ-4-002.

B. Gatekeeper's Cross-Motion for Summary Judgment

Gatekeeper filed a cross-motion for summary judgment, arguing that WZ-4-002 was not conceived or reduced to practice as part of a Novartis-funded research project, and therefore Novartis was not entitled to a license pursuant to the Research Agreement. For purposes of Gatekeeper's motion for summary judgment, I take the facts in the light most favorable to Novartis.

As discussed above, the only issue on summary judgment is whether Novartis was entitled to a license of the '419 patent. Even when the facts are taken in the light most favorable to Novartis, it cannot show that it was entitled to a license under the Research Agreement, the Consulting Agreements, or the Material Transfer Agreement with Dr. Eck. Therefore, summary judgment as to the issue of "entitlement" is appropriate for Gatekeeper.

IV. CONCLUSION

For the reasons set forth more fully above, I have denied the Novartis third-party defendants' motion for summary judgment (Dkt. No. 137), and granted Gatekeeper's cross-motion for summary judgment (Dkt. No. 144). The parties shall file on or before October 25, 2012 a proposed joint schedule for resolution of

the remaining issue of damages. A scheduling and status conference is set for 2:30 p.m., October 31, 2012 in Courtroom 1.

/s/ Douglas P. Woodlock
DOUGLAS P. WOODLOCK
UNITED STATES DISTRICT JUDGE